

74/F  
FLORA W. PATTERSON.

Mississippi Agricultural Experiment Station  
Agricultural College, Mississippi

INDEXED. PATH. COLL.

---

TECHNICAL BULLETIN No. 6

---

Forage Poisoning Due to *Claviceps*  
*Paspali* on *Paspalum* X

---

By  
H. B. BROWN, Ph. D.  
AND  
E. M. RANCK, V. M. D.

---



AGRICULTURAL COLLEGE, MISSISSIPPI

FEBRUARY, 1915

TELL FARMER, PRINTER, MERIDIAN

# STATION STAFF

---

G. R. HIGHTOWER .....	President
E. R. LLOYD .....	Director and Animal Husbandman
J. R. RICKS .....	Vice-Director and Agronomist
W. F. HAND .....	Chemist
W. N. LOGAN .....	Geologist
J. S. MOORE .....	Dairy Husbandman
A. B. McKAY .....	Horticulturist
R. W. HARNED .....	Entomologist
DANIELS SCOATES .....	Agricultural Engineer
H. B. BROWN .....	Cotton Breeding
J. M. BEAL .....	Botanist
E. M. RANCK .....	Veterinarian
C. F. BRISCOE .....	Bacteriologist
E. P. CLAYTON .....	Poultryman
S. S. JERDAN .....	Assistant Animal Husbandman*
N. F. HANSON .....	Assistant Animal Husbandman*
C. E. WILSON .....	Assistant Entomologist
MISS SIDNEY GAY .....	Stenographer
D. W. McILWAIN .....	Superintendent of Farm
J. C. KEAN .....	Foreman in Mule Breeding
M. D. REED .....	Assistant in Agronomy
E. B. FERRIS .....	Assistant Director, McNeill Station
C. T. AMES .....	Assistant Director, Holly Springs Station
G. B. WALKER .....	Assistant Director, Delta Station

---

\* In co-operation with U. S. Department of Agriculture.

# FORAGE POISONING DUE TO CLAVICEPS PASPALI ON PASPALUM

By H. B. BROWN, Ph. D. and E. M. RANCK, V. M. D.

Within recent years *Paspalum dilatatum* Poir., known commonly as "Paspalum", or "Large Water Grass", (fig. 1) has come into prominence as a forage grass, and has become rather widespread throughout many of the warm temperate regions of the world. It was probably introduced in the Southern States from South America,



FIG. 1.—A healthy head of *Paspalum dilatatum*. About 1-2 natural size.

but is now so widely distributed in these states from South Carolina to Louisiana that it is regarded as a native grass by many.

This grass was observed growing wild in the region of Agricultural College, Mississippi, about twenty years ago, and as it appeared to have certain desirable qualities, some effort was made to propa-

gate and distribute it. Now it is found growing very generally in this locality, and during the last three years specimens have been sent to the Experiment Station for identification from nearly every section of the State except the northeastern. In this State it is now used principally as a pasture grass, and it is recommended by the Experiment Station as a good general purpose pasture grass. It will grow on a heavy moist soil, and will also make fairly good growth on clay uplands during hot, dry summer weather; it seems to stand drought better than most grasses. During open winters it stays green, affording considerable pasturage. In some parts of the South it is cut for hay, yielding two crops of fair hay. Tracy (1) says, "It (*Paspalum dilatatum*) is a perennial which makes a continuous growth during warm weather, is not injured by close grazing or by moderate frosts, and so has a great value as a pasture plant for both summer and winter. . . . This grass is one of the best winter grazing grasses in the South, and it is well worth while to establish it on every farm south of Tennessee and North Carolina." Rolfs (2) says that *Paspalum dilatatum* proved the most satisfactory of the grasses tried in a forage grass experiment in Florida.

Wickson (3) found it to be a satisfactory grass for dry soils in California. Thompson (4), speaking of this grass in Guam (an island in the Pacific Ocean), said, "*Paspalum dilatatum* stood the drought well without irrigation and continued to give good results." Ritzman (5) says, in the Porto Rico Station report for 1912, "*Paspalum dilatatum* has been successful on low lands." In the Rhodesia (South Africa) Agricultural Journal (6) *Paspalum dilatatum* is mentioned as being a desirable summer pasture grass, having given good results on dry lands in that country. Williams (7), in New South Wales, Australia, says: "The grass is highly recommended for permanent pastures and is considered one of the best for general purposes. It has proved itself a mainstay for stock, growing vigorously when the fierce heat had withered up the other grasses." Spillman (8), in his discussion of important forage grasses in Bailey's Cyc. of Agr., says that in eastern Australia *Paspalum dilatatum* is by far the most important of the grasses; that it grows 5 to 6 feet high, remains green all the year, and is mostly used for pasture.

From the foregoing it will be seen that "*Paspalum*" is valued highly in many places. In nutrient elements it is somewhat below most of our forage grasses, yet it is fairly nutritious. A chemical analysis made at the State Chemical Laboratory, Agricultural



College, Miss., shows this grass to contain 33.6% crude fiber; 6.7% protein; 2.9% fat; and 48.7% carbohydrates. This is an average of three analyses.

There are about one hundred and fifty species of *Paspalum*, most of which are found in tropical and subtropical America. Small (9) lists fifty-five wild species found in Southeastern United States. Seventeen wild species have been collected in Mississippi. Although there are numerous species of *Paspalum*, there are but few of known economic value; the two most important are *P. dilatatum* and *P. platycaule*.

#### UNDESIRABLE FEATURES OF "PASPALUM"

From what has been said the reader may be inclined to conclude that *Paspalum dilatatum* is a very desirable grass. However, there are some undesirable features connected with it. If it once gets a start in a lawn or in a grass plot where it is not wanted, it is very hard to eradicate except by cultivation. Being a coarse grass, it is not attractive for lawns, and the heads are usually covered with "Honey-dew", a sticky exudation that clings to a person's clothing in a very annoying manner.

Some four or five years ago Prof. Lloyd, and certain farmers in the neighborhood of Agricultural College, began to suspect "*Paspalum*" as being poisonous to stock, especially to cattle. They noticed that cattle running in pastures where there was a goodly amount of this grass were often affected with a peculiar nervousness somewhat like that shown in certain stages of rabies. More recently the cases of apparent poisoning became so numerous that it was thought well to investigate the matter experimentally. In September, 1914, the writers began the study. A quantity of the grass was cut and fed to calves, and in a few days the characteristic symptoms of poisoning appeared. This was taken as an indication that the suspicion was well grounded, and a careful study was started.

#### FUNGI INFECTING "PASPALUM"

A microscopical examination of the heads of the grass showed that a large per cent., probably eighty or ninety, were diseased. Three different species of fungi were found infecting them, *Claviceps Paspali* Stevens and Hall, *Fusarium heterosporum* Nees., and a *Cladosporium*.

The *Claviceps* seemed to appear first, for it was found on the youngest heads; it showed, in its first stage, in ten days or two weeks

after the heads first appeared. This fungus attacks the pistils or female flowers and grows as a parasite in them. Soon there is produced a mycelium, or mass of fungus threads that occupy the space between the glumes of the spikelet. This mass of fungus tissue produces in a short time numerous conidia, or spores, and a sticky exudation, or "Honey-dew", which carries the conidia from the plant. These conidia are single celled, about  $5\mu$  wide and  $15\mu$  long, oblong, hyaline, with contents homogeneous except for a cluster of granules in each end. (See fig. 2.) This stage in the life history of the fungus is known as the sphacelial stage. In this stage spores are produced in great abundance, and much "Honey-dew" exuded. It is also principally in this stage that the fungus is carried from plant to plant,



FIG. 2.—Sphacelial spores from the "Honey-dew" of *Claviceps* on *Paspalum dilatatum*. Magnified about 720 diameters.

scattering the disease. Bees, bugs, flies, beetles, and insects of many other kinds visit the diseased heads to gather the sweetish "Honey-dew"; some of this sticks to their bodies, and when they fly to healthy heads, spores are carried in the exudation clinging to their bodies, and new infection takes place. The spores may be scattered also by cattle, or by any animal walking through the grass, or by the wind causing the heads to move about and strike together. Mercier (10) reports that the conidia of one species of *Claviceps* are carried on the hair and in the alimentary canal of a fly, *Sciara thomae*.

Some days after infection takes place, the mass of fungus threads between the spikelets, mentioned above, enlarges and forms a rather dense globular mass, 2 mm. to 4 mm. in diameter, known as a sclerotium. This fungus nodule, or sclerotium, is yellowish to grayish in color, and

more or less roughened and broken on the outside when mature. (See fig. 3.) A section of a sclerotium shows it to be nearly homogeneous in structure, and to contain a considerable quantity of oil. The sclerotia remain attached to the heads until some time during the winter, falling to the ground at the time the old grass head goes to pieces and sheds its spikelets. They lie on the ground for some months in a resting condition. Later new growth starts, and there is sent out from each sclerotium one or more slender stalks, or stipes, a few millimeters in length, each bearing at the outer end a dull yellow head about 1 mm. in diameter. (See fig. 4.) Imbedded in the head

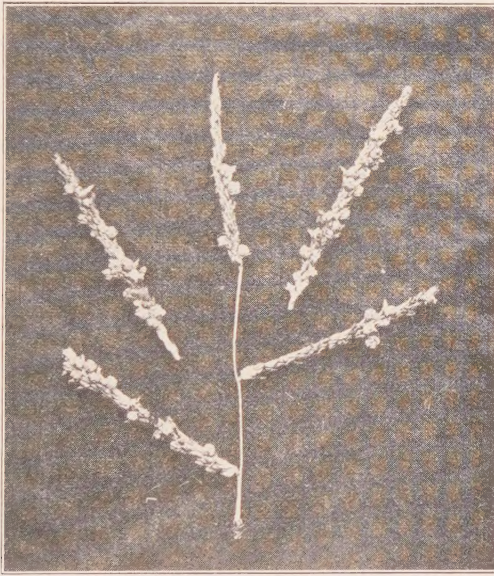


FIG. 3.—Diseased spikes of *Paspalum dilatatum* showing sclerotia, or fungus nodules. About 1-2 natural size.

are numerous small perithecia, each containing asci and ascospores. The ascospores are filiform, about  $1\mu$  in diameter and  $60\mu$  to  $70\mu$  long.

Sclerotia gathered in the field February 15 produced stipes and heads in the laboratory by March 27, and ripe spores were observed April 15. (The sclerotia were germinated on moist filter paper in petri dishes.) Stevens and Hall (11) report that sclerotia of this species collected from the ground in the spring produced stalks in the laboratory in 20 to 25 days. They report further that



Rolfs saw the ascigerous stage of *Claviceps Paspali* in 1901, and that he noticed that the ascospores were disseminated by beetles, principally of the family Carabidae, which, running over the ground, come in contact with the spores, and later, in seeking a high point from which to fly, run up the grass culms and over the pistils, thus bringing about infection.

*Claviceps purpurea* (Fr.) Tul., or ergot, a species which grows on rye and other grasses, produces sclerotia that have been observed by Zimmerman (12) to begin growth and produce asci about the time the rye is in flower. Falck (13) says that the ascospores of *Claviceps purpurea* may be carried by the wind and produce infection. Stäger (14) claims to have demonstrated by experiments that summer spores of *Claviceps purpurea* which live over winter still possess (when



FIG. 4.—Sclerotium of *Claviceps Paspali* producing reproductive parts after lying on the ground all winter. Enlarged about four diameters.

ten months old) their full power of germination and infection. Probably in the case of *Claviceps Paspali* the first infection of the season is brought about by means of ascospores, which are carried to heads in flower. After a few heads are infected, conidia are produced abundantly and scattered so freely that the disease spreads rapidly.

#### SPECIES OF CLAVICEPS

While there are undoubtedly several species of *Claviceps*, it is impossible to say how many, on account of the fact that one species may grow on a number of different hosts, and on the different hosts may vary considerably. *Claviceps purpurea*, for instance, is said by Barnes (15) to grow on thirty-five different grasses. Stäger (16) succeeded in inoculating rye, *Anthoxanthum odoratum*, *Arrhenatherum elatius*, *Phalaris arundinacea*, *Poa pratensis*, *P. alpina*, *P. sudetica*, *P. hybrida*, *P. caesia*, *Hierochloa borealis*, *Bromus sterilis*, *Dactylis glomerata*, *Hordeum murinum*, barley, *Briza media*, and *Calamagrostis arundinacea* with ergot from rye. Heald and Peters (17), in 1906, reported *Claviceps purpurea* as growing on nine species of wild wheat grasses and on eight species of wild rye grasses in Nebraska. Wil-



liams (18), in 1893, reported ergot common on wheat grasses throughout South Dakota, and he mentioned in particular *Elymus* sp., *Calamagrostis canadensis*, *C. confinis*, *Phalaris arundinacea*, *Poa pratensis*, *P. compressa*, *Agropyrum glaucum*, and *A. tenerum*. Stevens and Hall (11) have found species of *Claviceps* on *Paspalum dilatatum*, *P. laeve*, and *Tripsacum dactyloides* in North Carolina. The writers have found *Claviceps Paspali* on *Paspalum dilatatum* and *P. distichum* in Mississippi. While this list is not at all exhaustive, it will serve to give some notion of the variety of hosts of species of *Claviceps*.

Stäger (16), in 1900, considered that there were five species of *Claviceps*: *C. purpurea*, *C. microcephala*, *C. Wilsoni*, *C. pusilla*, and *C. setuosa*. Since that time several others have been described.

## POISONOUS PROPERTIES OF SPECIES OF CLAVICEPS

### HISTORICAL

It has long been known that the *Claviceps* or ergot of grain, especially rye, possesses certain poisonous properties, and that it will affect both man and animals. Wood states that epidemics of ergotism, or chronic ergot poisoning, have been recorded from time to time since the days of Caesar (B. C. 190-44). There is much reason for doubt, however, in regard to the diagnosis of cases occurring before the tenth century. According to Pammel (19), epidemics were frequent in France from the ninth to the thirteenth century, and in Spain in the twelfth. They were first called plagues, and prayers were offered for their removal, but later they came to be known under other names. In 1596 Hesse and adjoining provinces were visited by a plague which was attributed to the presence of ergot in grain. In the epidemic in Silesia in 1722, the king of Prussia ordered an exchange of sound rye for the affected grain. Freiburg was visited in 1702, Switzerland in 1715-16, Saxony in 1716, and other districts of Germany in 1717, 1736, and 1741-42. France was visited in 1650, 1670, and 1674. From 1765 to 1769 ergot was abundant in rye and barley in Sweden. In 1816 an outbreak of ergotism occurred in France, which was especially fatal to the poorer inhabitants.

It has been observed that these epidemics follow a rainy season. Fleming states that in 1041, when the weather was so unpropitious—tempests, rains, and inundations occurring—many cattle perished from the disease. In 1608, after inundations and heavy fogs, there was a general epizootic among the cattle in Germany. In the same year ergotism appeared in the human species.

Dr. Randall, in 1849, called attention to the disease among cattle in New York, in which the involved parts were finally invariably affected with a dry gangrene. He stated that in the severe climate of New York farmers allowed their cattle to winter in fields of blue grass, *Poa pratensis*, which was rich in ergot. A disease known as "hoof-ail" was correctly ascribed to ergot by James Mease, of Philadelphia, prior to 1838. The disease was quite severe in Orange county, New York, in 1820. It was minutely described by Arnell. In 1857 the disease was quite severe in Portage county, Ohio. A committee appointed by the Farmers' Association of Edinburg reported that the disease was due to ergot contained in the hay eaten by cattle. In recent years epizootics of ergotism have been reported by Law in New York, Stalker in Iowa, and Faville in Colorado. Harshberger has called attention to an outbreak of ergotism from the use of ergotized red top, *Agrostis alba*, the fungus being common on red top throughout the United States, and one of the most common impurities in red top seed.

In 1884, following serious outbreaks of ergotism in Kansas, Missouri, and Illinois, appeared Salmon's (20) classical work on ergotism. In this work the symptoms and characteristics of the disease are described by Dr. Salmon as follows: "The first symptoms of disease in the Illinois epizootic were diarrhoea, lameness, stiffness of the lower joints of the affected limbs, and coldness and insensibility of the same parts. In Kansas this derangement of the digestive apparatus was not noticed. At all the places visited, however, the lesions of the feet were of a common character and were produced by a common process. In the more severe cases a constricted band formed around the limb at the point separating the gangrenous from the living flesh. So marked was this constriction that some of the owners looked upon it as the initial lesion of the disease and cut across it with a knife in the hope of re-establishing the circulation. It is needless to say that this hope was delusive, since the part below the constriction was entirely lifeless before this was formed. The constriction was the first step in the effort of nature to rid the body of parts that were of no further use to it.

"The next in the process of separation was a crack in the skin at the upper edge of the band of constriction, which gradually extended toward the center of the limb, the softer parts dividing first, and the tendons and ligaments resisting much longer. Generally this separation was in the vicinity of a joint, and in this case, as the

lower members of the limb were lost, a comparatively even surface was left which healed readily. Some animals lost only a toe, the dividing line passing through the joint between the *os pedis* and *os coronae*; others lost both the *os pedis* and *os coronae*; still others lost the three lower bones, and the line of separation passed through the fetlock joint, while in the most severe cases the line of constriction formed at the upper third of the metatarsal bone and the fleshy parts sloughed off, leaving the uncovered bone protruding for more than half its length. The gangrene was not confined to the feet, for there were individual animals which were losing from 2 to 6 inches of the lower part of the tail. . . . In a few cases a part of the ear was found in the same condition.

“One of the most interesting features of the enzootic . . . was the implication of the mucous membrane of the mouth. With some animals, this was limited to a more or less diffuse discoloration, without the loss of substance. More frequently there were circumscribed dark red spots or patches, from a fourth to an inch in diameter. Very often there was a loss of substance-erosions from a third to a half inch in diameter. . . . In a very few animals a lesion of a different character was observed in the mouth. In these cases an irregular patch of mucous membrane from 1 to 2 inches in diameter was elevated, corrugated upon its surface, hard, insensible, and of a light color, tinged with pink and yellow. It seemed to be a circumscribed gangrene of the mucous membrane, the dead parts being partially decolorized by soaking in the fluids of the mouth.

“There was also an evident irritation of the mucous membrane of the posterior parts of the alimentary canal and organs of generation. That covering the rectum and vagina was generally red, covered with mucous, and presented spots denuded of the epithelium. In Missouri six cases of abortion in cows were reported, and in Illinois there were many cases of abortion and difficult parturition with mares.

“The constitutional symptoms were not very marked. The temperature of the animals which I examined was about normal, with the exception of a few from which one or more limbs were sloughing and with which there was suspicion of septic poisoning.”

In all the cases mentioned above, Dr. Salmon found upon investigation that the cattle had eaten a considerable quantity of ergot with their forage, the amount of ergot on wild rye and on some



other grasses being unusually great that year. He found further that the ergot was more likely to cause trouble in extremely cold weather, especially if the cattle were not fed good nutritious food and furnished plenty of water.

In more recent years fewer outbreaks of ergotism have occurred, due doubtless to a better understanding of the poisonous nature of the fungus. Stalker (21) reported a disastrous outbreak in Iowa in 1892, and in 1899 Wilcox (22) reported cases of ergot poisoning among horses in Montana. The horses were afflicted with first a partial, and finally a total, paralysis ending in death usually in 6 to 12 hours.

Practically all of the domestic animals seem to be susceptible to ergot poisoning. Cases, either accidental or experimental, have been reported in mules, horses, cattle, sheep, hogs, dogs, cats, and fowls; man also is susceptible.

The cases of ergotism that have been mentioned were all due, probably, either to *Claviceps purpurea* or to some closely related species. The symptoms of *Claviceps Paspali* poisoning are very different from the foregoing, as will be pointed out later.

#### CHEMISTRY OF ERGOT

Although ergot, principally *Claviceps purpurea*, has been studied by a number of able chemists, it cannot be said as yet that its chemistry is well understood. A study of the chemical composition of *Claviceps Paspali* is now being made by Dr. W. F. Hand, of the Chemical Department of Mississippi Agricultural and Mechanical College.

#### FUSARIUM HETEROSPORUM

The second fungus found on the heads of *Paspalum dilatatum* was a *Fusarium* which has been identified by Tracy (23) as *Fusarium heterosporum* Nees. This species has also been mentioned by Massee (24) as attacking the grain of wheat, rye, barley, and various grasses, as *Holeus*, *Lolium*, *Paspalum*, *Panicum*, etc.

In so far as our observations here in Mississippi go, this fungus attacks only the *Paspalum* heads that are infected with *Claviceps*. Probably the "Honey-dew" serves to catch the *Fusarium* spores, and also serves as a good medium in which they may germinate and grow. The fact that this species of *Fusarium* is so commonly closely associated with species of *Claviceps* has led some authorities to consider it simply a conidial stage of *Claviceps purpurea*; Ellis and Everhart (25). Cultural evidence does not indicate any relationship.

In attacking a head of "Paspalum" this fungus first forms a mass of white hyphae between the spikelets, crowding them apart. Soon the surface of this mass is covered with conidia, which in mass are a brilliant orange-red color. The conidia are fusiform, slightly curved, 3-6 septate,  $27-73\mu$  long, and  $5-9\mu$  wide. (See fig. 6.) A little



FIG. 6.—a. Macrospores of *Fusarium heterosporum*.  
b. Macrospores germinating.



FIG. 7.—a. Mycelium of *Cladosporium*.  
b. Conidiophore.  
c. Spores.  
d. Germinating spores.

later the hyphae run from one spikelet to another over portions of the spike, thus gluing them together and obscuring them more or less.

#### POISONOUS PROPERTIES OF FUSARIUMS

Various species of *Fusarium* have been suspected as being poisonous when producing mouldy corn, fodder, etc., but the evidence is somewhat contradictory. The toxic effects probably vary greatly with conditions. Horses, cattle, sheep, dogs, and cats are sometimes affected with a form of meningitis due to mouldy foods. Dr. Butler and Dr. Mayo at the Agricultural College, Manhattan, Kansas, in an experiment with mouldy corn, fed two colts, twenty three months old, about three kilos daily (about 6.6 lbs.). One colt died at the end of thirty-six days. In another experiment, a two-year

old colt was fed with mouldy corn and good prairie hay. This colt died at the end of twenty-seven days.

Pammel (19), quoting from Dr. Low, says: "Fodders affected with cryptogams or bacterial ferments are undoubtedly at times the cause of encephalitis. Veterinary records furnish many instances of widespread attacks of stomach staggers, abdominal vertigo, and cerebro-spinal meningitis in wet seasons, when the fodders have been harvested in poor condition or when from inundation or accidental exposure they have become permeated by cryptogams and microbes.

. . . The experimental administration of moulds, smuts, and microbes, have in a great majority of cases led to little or no evil result (Gamgee, Mayo, Dinwiddie, etc.), and there is a strong tendency to discredit the pathogenic action of these agents in reported outbreaks. The safer conclusion, perhaps, would be to recognize the fact that they are not equally pathogenic under all conditions of their growth and administration."

*Fusarium heterosporum* growing on *Paspalum dilatatum* appeared to have no poisonous properties, as shown by feeding experiments to be mentioned later.

#### CLADOSPORIUM

The third fungus found on the heads of *Paspalum dilatatum* has not yet been identified definitely, but it is probably a species of *Cladosporium*. With further study we hope to identify it definitely and to learn more of its life history. At present we can say only that it appears to attack old heads, or ones already badly infected with the other fungi mentioned. The mycelium is made up of branching, dark colored, septate hyphae, about  $3.5\mu$  in diameter. This spreads over the surface of the whole head. Conidiophores over the surface of the mass of hyphae bear numerous small, dark, two-celled spores,  $10-17\mu$  long and  $5-7\mu$  wide. (See fig. 7.)

This fungus is not as common as the others mentioned, and our feeding tests, to be mentioned later, showed no indication of poisonous properties.

#### CULTURES

Sphacelial spores from the "Honey-dew" of *Claviceps Paspali* and conidia from *Fusarium heterosporum* were placed in hanging drop cultures, both in water and in a weak sugar solution. *Cladosporium* spores were placed only in water. The sphacelial spores



failed to germinate. The other spores germinated freely in both media in a few hours. (See figs. 2, 6, and 7.) The *Cladosporium* spores germinated after being kept in the laboratory from October until February.

On October 24, 1914 an attempt was made to get pure cultures of the *Claviceps* and *Fusarium*. Bean pods, peptone agar, and *Paspalum* heads were used as media. These were inoculated with *Fusarium* conidia, sphacelial spores, and pieces of *Claviceps* sclerotia. The *Fusarium* spores germinated freely, and several pure cultures



FIG. 8.—1. *Fusarium heterosporum* growing on peptone-agar in culture.  
2. *Fusarium heterosporum* growing on sterile *Paspalum* heads in culture.  
3. *Claviceps Paspali* growing on sterile bean pods in culture.

of this fungus were easily obtained. (See fig. 8.—1. and 2.) Some of the pieces of sclerotia produced growth, and three pure cultures of the *Claviceps* were obtained. (See fig. 8.—3.) These were all on bean pods. The fungus grew slowly throughout the pod, and up to the present has shown no signs of producing reproductive bodies. The *Fusarium* cultures grew rapidly on all three media, and produced

an abundance of white, cottony mycelium. Spores were noticed on November 19.

## FEEDING TESTS

### GUINEA PIGS

On December 11, 1914 guinea pig No. 1, a pig weighing 350 grams, was fed 1.9 grams of *Fusarium heterosporum* from pure culture. The living fungus was peeled from bean pods with forceps and put into the pig's mouth. The pig chewed and swallowed it rather freely. On December 12, 1914, two grams were given; December 13, 1914, four grams; December 14, 15, and 16, 1914, two grams



FIG. 9.—Guinea pig No. 2 in normal condition.

each day; and on December 18, 1914, four grams. The pig remained perfectly normal during this feeding. The same pig was then fed daily three or four grams of dried *Paspalum* heads badly infected with *Fusarium*. This feeding was continued four days. Pig remained normal.

On December 26, 1914 guinea pig No. 2, a pig weighing 300 grams, was fed twenty-five *Claviceps sclerotia* picked from diseased "*Paspalum*" heads. (See fig. 3.) They were thoroughly dry, having been in the laboratory since October, and weighed .17 grams. (Twenty-five of the larger sclerotia weigh .2 grams, but all sizes were used in the feeding experiments.) The sclerotia were put into the pig's mouth, one by one, with forceps, and the pig held until it had chewed

and swallowed them. By this means it was possible to tell definitely just how much of the fungus the animal received. No effects were noticeable at 11 a. m. December 27, 1914, when another dose of twenty-five sclerotia was given. During the afternoon of the 27th, the pig seemed somewhat nervous and was trembling slightly. At 9 a. m. the following day, the 28th, it was decidedly nervous and trembled violently when excited by the noise of a hand clap, etc.; it was beginning to show exactly the same symptoms that the affected cattle showed. At 10 a. m. December 28, it was given a dose of



FIG. 10.—Guinea pig No. 2. under the influence of *Claviceps Paspali* poison.

fifty sclerotia. At 4 p. m. the same day, it was trembling and shaking more violently. At 7 a. m. December 29, pig was down and could not get up; legs were stretched out straight, were stiff, and trembling; eyes bulged out: at 11:30 a. m. symptoms seemed to be about the same: at 4 p. m. the pig appeared much better, was on its feet, could walk with a staggering gait, and did not tremble much unless excited by a shock; it also tried to eat corn. No more of the fungus was fed this pig for some days. On January 2 it seemed to be in a normal condition. The tests just mentioned seem to show that the poisonous element is in the sclerotia, since the pig received no other food except corn; that the fungus retains its virulence in a dry state, for the sclerotia had been in the laboratory for



several weeks; and that the effects are due to a poison, since the animal soon recovers after it ceases to receive a supply of the sclerotia.

On January 7, 1915, we again began feeding sclerotia to pig No. 2. (See fig. 9.) It was fed twenty-five *Claviceps sclerotia* on January 7 and 8. On the 9th it was fed fifty sclerotia. No particular effect was noted that day, but on January 10 the pig was shaking and trembling considerably. It was then given another dose of fifty sclerotia. At 8 a. m. January 11, it was shaking a good deal but was not in a serious condition; it was able to scamper about, and ate corn, though it had some trouble in getting hold of it. At 10 a. m., one hun-



FIG. 11.—Guinea pig No. 2 killed by *Claviceps Paspali* poison.

dred sclerotia were fed; the same dose was given each day until January 15. At 9 a. m. on January 12, the pig was shaking a little worse, perhaps, and could scarcely eat, but could stand up. At 9 a. m. January 13, it was shaking more violently; could stand up, but could scarcely walk or eat. At 11 a. m. January 14, it seemed to be trembling about as before (fig. 10), but at times would take more violent spells, tumble over, and turn somersaults. At 8 a. m. January 15, the pig was found dead. (See fig. 11.) It then weighed 260 grams, having lost 40 grams since December 26.

The second experiment with pig No. 2, just discussed, was to see if more of the fungus would be required to produce poisoning the second time; or, in other words, to see if the animal developed any resistance to the poison. In the first experiment the pig became de-

cidedly nervous after being fed fifty sclerotia, while in the second, not until after it had been fed one hundred. In the first it became prostrate after being fed one hundred sclerotia, but in the second it was fed five hundred and fifty before becoming prostrate.

Guinea pig No. 3, a large pig weighing 491 grams, was fed as follows: January 11, forty sclerotia (this pig was given a larger dose on account of its large size, forty for it being equivalent to twenty-five for a smaller animal); January 12, forty sclerotia; January 13, eighty sclerotia; January 14, one hundred and sixty sclerotia. The pig seemed to be trembling slightly three and one-half hours after being given the first dose; its nervousness increased rather gradually, reaching a climax on January 15. It was fed only regular feed (cracked corn and cabbage) on the 15th, but after eating became more nervous and had tumbling spells—would throw head back as if it were being pulled backward, and then rear up and fall backwards. Later in the day the pig seemed improved, and was much better the following day, but still trembled considerably. It was fed only regular feed for a few days, and by January 18 had reached a normal condition. On January 22, treatment was again started and pig was fed as follows: January 22, forty sclerotia; January 23, forty sclerotia; January 24, eighty sclerotia; and January 25, one hundred and sixty sclerotia. Pig died after this last feeding; it was probably injured in the feeding process. This pig became nervous a little sooner during the first treatment, but the test was not satisfactory, due to the injury causing death.

Guinea pig No. 4, a small pig weighing 195 grams, was fed twenty-five sclerotia at 3 p. m. February 10. At 9 a. m. next day, the pig was shaking considerably and seemed to be rather sick. It was fed no more of the fungus, but continued sick and died February 15. The pig was not in a vigorous condition when the experiment was started.

Guinea pig No. 5, a medium sized pig, was fed a gram of extract made from "Paspalum" heads infected with *Claviceps Paspali*. Just after the dose was administered the pig appeared sluggish; in an hour it was trembling violently; in four hours it was shaking still more and could scarcely stand up—stood with legs spread apart; in ten hours it was very sluggish; and in nineteen hours it was dead.

Guinea pig No. 6, an old female pig, was fed a gram of the extract, like that mentioned above, at 10:30 a. m. Symptoms were

very similar to those of pig No. 5. Pig was found dead at 9 a. m. next day. A count of the red blood corpuscles was made before giving the first dose, then again at 4 p. m. and 8 p. m. No difference could be detected.

A cat fed meat having on it some of the extract mentioned above, soon developed the characteristic symptoms.

Guinea pigs 7, 8, and 9 were given *Claviceps Paspali* extract made in different ways. All died within twenty four hours, showing

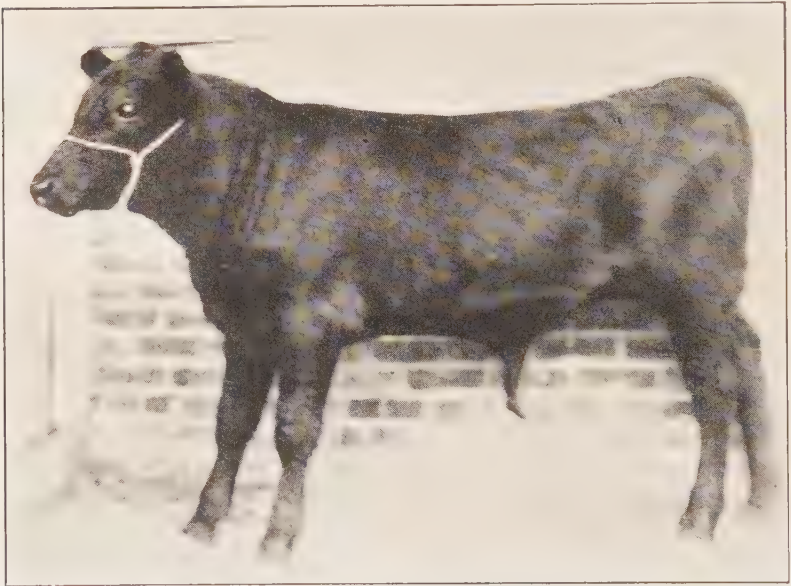


FIG. 12.—Calf No. 1.—This calf was sent to the Veterinary Hospital for treatment. It showed symptoms of "Paspalum" poisoning after being on "Paspalum" pasture but four days. At the hospital it was fed good, sound hay and some grain; the nervousness disappeared in about a week.

symptoms similar to experimental animal, guinea pig No. 5. Abortion occurred in one of these. This was the only pregnant pig that was used in the experiment in so far as is known.

The guinea pigs that were fed sclerotia ate them rather freely in most cases; some seemed to have a ravenous appetite for cracked corn, their regular feed, after being fed sclerotia. It was noticed, too,



that frequently just after being given a dose of the sclerotia, the animal drank a good deal of water. The sclerotia have a somewhat pungent taste; probably this led to the desire for water. The animals urinate freely and bowels are always loose in advanced stages of the poisoning.

Guinea pig No. 10, a pig weighing 203 grams, was fed twenty-five pieces of "Paspalum" heads, each piece consisting of three or four spikelets glued together by dried "Honey-dew" of the Clavi-

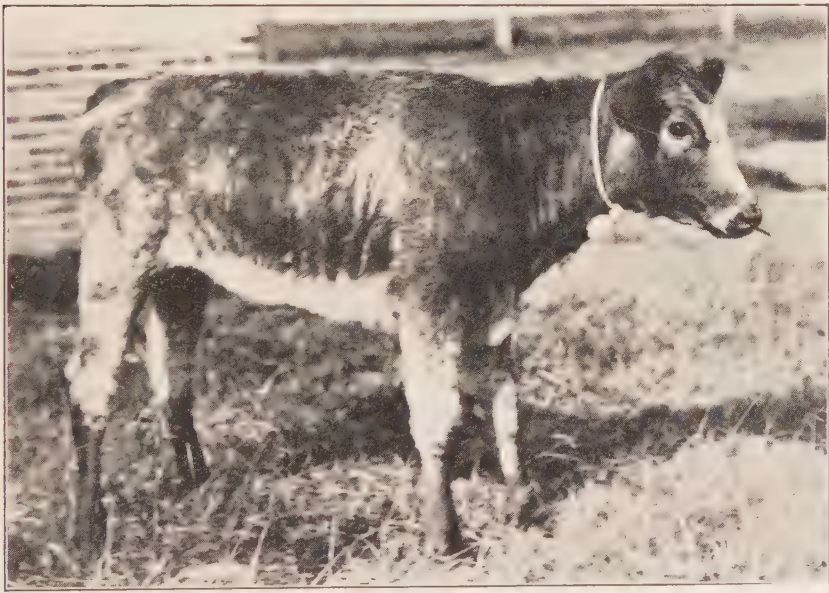


FIG. 13—Calf No. 2 after being fed diseased "Paspalum" heads some days.  
Note stiff appearance with legs rather wide apart to assist in standing

ceps, and in most cases the pieces were more or less covered with a black mould, the Cladosporium, or third fungus mentioned above. The next day the pig seemed to be normal and was fed about forty pieces like the ones just mentioned. The same amount was continued for five days. The pig showed no effects during the feeding nor afterward. Apparently the Cladosporium and material produced in the sphaelial stage are not poisonous, at least not after drying.

Guinea pig No. 11, a half grown pig weighing 200 grams, was

fed extract of commercial ergot (*Claviceps purpurea*) as follows: First day, 1-4 c. c.; second day, 3-8 c. c.; third day, 3-8 c. c.; fourth day, 1-2 c. c.; and 1-2 c. c. each day for four days. This treatment produced no nervousness or trembling such as *Claviceps Paspali* produces. In fact, the effect seemed to be slight although, toward the end



FIG. 14.—Calf No. 2. This picture shows animal in stage of excitement during which it cannot remain on its feet. Note peculiar expression of eyes, also the chin resting on the ground to help maintain position. All that was necessary to cause the animal to assume this position was to clap the hands and jump toward it. The nervous paroxysm would immediately come on and last one or two minutes.

of the experiment, the pig became more sluggish and moved about slowly; this indisposition lasted for some days after the ergot was withdrawn.

#### FEEDING TESTS WITH CALVES

On September 24, 1914 we began feeding "Paspalum" to two grade calves obtained from the beef cattle department of the Experiment Station. One animal was fed on heads of "Paspalum" grass free from disease, and the other was fed heads from infected plants. On September 26, or two days after starting the experiment, the calf re-

ceiving the diseased heads became nervous, and shook as though suffering with a slight chill. Each day the animal showed more extensive nervousness; there was a wild expression from the eyes, fig. 12; and the ears were held erect as if listening for something, fig. 13. Upon the slightest noise or excitement, such as clapping hands, the animal would show such extreme nervousness that it could not stand on its feet. This nervousness was noticed especially to affect the control of the front feet, fig. 14. As the experiment progressed and the feeding of the poisoned heads continued, the animal became more violently nervous. One peculiarity noticed in the feeding was that the animal seemed very fond of the diseased plants. This peculiarity has been observed also by others, animals grazing in "Paspalum" pastures apparently selecting the diseased heads and eating them in preference to the sound plants.

The amount of material fed each day varied somewhat, as our custom was to give the animal all it would clean up, and to simulate pasture conditions as nearly as possible. It was observed that the animals on pasture seemed to clip off the heads rather than eat the whole plant. This led us to use later the diseased heads alone. In handling the heads many fungus nodules, or sclerotia, dropped off; these were all collected, mixed with the grain food, and fed in a flat trough. The animals seemed to prefer this material to that contained on the whole plant. (The grain food comprised equal parts of shorts, bran, and corn chops.)

The temperature of the calf was taken each day, morning and evening. No elevation was noted. The range of temperature was 100.5° to 102.5° F. The pulse rate, however, was enormously exaggerated, especially during one of the nervous paroxysms when the animal was prostrated. The heart beat could be felt and heard through the chest wall when the front leg was pulled forward. Just after the animal fell, the beat would frequently go as high as 180 or over, and then gradually slow down in a few moments to about 100 per minute. It was impossible to determine if the heart ever assumed nearly the normal number of beats per minute for the reason that just as soon as

we approached the animal, the nervousness was so pronounced that the beat was at once accelerated.

The material fed had apparently no peculiar effect upon the eliminations. Bowel and kidney actions were, to all appearances, normal. No observation has been recorded indicating that the material was in the least irritating, or that it produced scours even if fed to young calves. The peculiar symptoms indicate, however, that the poisoning is of a cumulative nature, and that the system is not



FIG. 15.—Calf No. 2. Note that the entire lower jaw, neck, and breast are used by the animal in maintaining this peculiar position. Note also that the eye is partially closed and drawn; this is very different from the first symptoms noticed.

able to eliminate the poison until a number of days have elapsed after the withdrawal of infected feed.

The symptoms of nervousness in the animal became more pronounced each day until October 11, or seventeen days after the experiment started, when it was in a very bad condition, fig. 15. This showed clearly that the poisoned grass was gradually producing very much



more intensified symptoms of poisoning. At this time the animal seemed uncomfortable while on its feet,—would lie down most of the time, and even preferred to eat while lying down. It was noticed that while attempting to drink water from a bucket, it would develop a paroxysm, lose control of its front legs and often plunge its head into the bucket and upset it. This extreme nervousness, noticed in cattle grazing on pastures in which the only source of drinking water is ponds, accounts for a great many animals being found dead at the



FIG. 16.—Calf No. 2 prostrate. Note drawn expression of eyes and slight drawing back of head (aposthotonus). In this condition the animal breathes rapidly, shows consciousness, and responds to noise by twitching.

edge of ponds with only a part of the head submerged in the water.

After October 20 the animal was unable to eat, drink, or hold its head in normal position, and assumed a prostrated position, such as is shown in fig. 16. It was kept in a box stall free from excessive light and noise on a soft and comfortable bed; attempts were made to feed and water without success until October 23. It was then plainly seen that death was a matter of only a few hours, and so the animal was destroyed.

**POST MORTEM**

Careful examination was made of all organs after death, and we were unable to locate anything of peculiar or interesting nature except in the kidneys and brain. The capsule of the kidneys was friable, and small echymotic spots were found in several lobes of each organ. Nothing unusual was noted on sectioning, and nothing found which would indicate organic lesions in the kidneys. The bladder was empty.

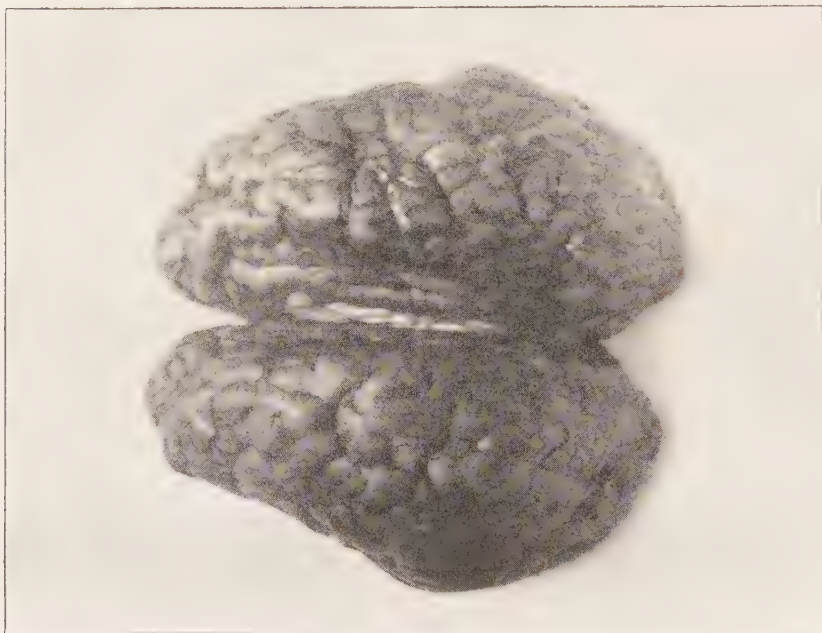


FIG. 17.—Brain of Calf No. 2. Note prominence of blood vessels over entire organ. This congestion was also noticed throughout all the coverings of the organ and extended to the cord.

In the cranial cavity was found an intense congestion of the entire brain and meninges. Every part of the organ and coverings showed prominent evidences of congestion. Blood vessels were much engorged and prominent. (See fig. 17.) A guinea pig was inoculated with the blood from this animal, and another with liquid from the brain cavity. Neither inoculation produced any effect.

During the time of feeding this calf the diseased heads, we also

fed another calf of approximately the same size, weight, and age, with heads of "Paspalum" free from disease. This animal showed not the slightest symptoms of poisoning. After the first, or poisoned, animal was destroyed, we then fed the second calf diseased plants. At the end of three days it showed symptoms identical with those exhibited by the first calf. Upon withdrawing the diseased plants for a week or more, the animal recovered without any treatment in the form of medicine.



FIG. 18.—A steer recovering from a prolonged attack of "Paspalum" poisoning.

#### TREATMENT

So far as treatment is concerned, we advise the administration of a regular dose of epsom or Glauber salts, and the withdrawal of affected animals from pastures containing diseased plants. We have known of many cattle affected with the trouble which recovered without any treatment whatever. We think, from our observations, that no quick results can be looked for from giving any medicine, since the poison seems to affect the nervous system and is gradually eliminated after the withdrawal from infected feed.

## GENERAL

As has been mentioned, most domestic animals are seriously affected by eating *Claviceps purpurea*, or ergot, if it is eaten freely with their forage feed. *Claviceps Paspali*, on the contrary, is known to affect only cattle running in pastures where the infected grass is growing. It is possible, though, that other animals are not affected because they avoid the diseased heads of grass, for in our experiments every animal that was fed the fungus promptly developed the characteristic symptoms.

Our experiment has not yet been carried far enough to determine whether or not animals develop resistance to the poison. Farmers are of the opinion that cattle will be affected the second time as quickly as the first, and our feeding experiment with calves seems to bear out this conclusion. But the experiment with guinea pigs seems to show that some resistance was developed, for in each case more of the poison was required to produce effects the second time it was administered. Cattle in pastures are apt to develop an abnormal appetite for the fungus and will wander about biting off simply the diseased heads; in this way they may get a large amount of the poison, but the exact amount obtained at any particular time cannot be determined. When feeding guinea pigs the sclerotia alone, it is possible to tell more definitely about the amount taken.

During the late summer and fall of 1914, cases of "Paspalum" poisoning were more numerous than ever before. On the College farm there were five cases among the dairy cattle, fifteen among the beef cattle, and three among the mess hall cattle. Cattlemen and farmers in the vicinity of this Station have also had a number of cases. One of the men says that he first noticed the disease five or six years ago, and that it has gradually become prevalent, having been considerably worse during 1914 than ever before. He says also that he has had twenty-five or thirty cases during the past season while the cattle were on grass, four or five of these cases fatal, and that he has had six cases during the present winter while feeding hay. Another reports that he was never bothered with "Paspalum" poisoning until



last summer. He had then about a hundred cases, two of which were fatal. Another states that he first noticed the disease in his herd about three years ago, that he had twenty-five or thirty cases last year, and that most of these were fatal. Very probably a number of the suspected cases of "Sneezeweed" poisoning occurring in different parts of the State are really "Paspalum" poisoning.

Cattle that die from "Paspalum" poisoning are not, in most cases, killed by the poison directly, but get down in the pasture under its influence and perish on account of lack of water and food. If they are found in the early stages and taken from the pasture so they cannot get any more of the fungus, and given plenty of food and water, they will recover in a few days. On the other hand, if they feed on the diseased grass for some time after getting under the influence of the poison, the effects will be more permanent. Professor Lloyd has observed that a steer that was under the influence of the poison last summer has failed to gain in weight when on good feed this winter. Figure 18 shows a steer that is just recovering from a prolonged attack of the poisoning.

#### CONTROL MEASURES

Any plan that will prevent the development of the *Claviceps* sclerotia will prevent poisoning. If a mower, set so as to clip off the "Paspalum" heads, is run over the pasture about once a month during August, September, and October, the trouble will be prevented. If the pasture is burned over during the winter, many sclerotia lying on the ground will be destroyed; this may lessen the disease, since the fungus winters over in the form of sclerotia and new infection takes place from them, but this plan cannot be favored very strongly for some sclerotia will not be destroyed; and infection, once started from them, will be spread rapidly by insects, etc. Then, too, the harm done the pasture by the burning may more than overbalance the good obtained.

In the case of *Claviceps purpurea* poisoning, epidemics have come at intervals; after a bad outbreak in a locality, there might not be another for many years. The spread of the fungus seems to

be controlled in part, at least, by nature. In a similar way, natural enemies may arise to check the spread of *Claviceps Paspali*. The other two fungi mentioned in the foregoing discussion, the *Fusarium* and *Cladosporium*, very probably prey upon the *Claviceps* and prevent its development and reproduction to a certain extent. However, natural means of control do not seem to be in the ascendancy as yet. We do not know that the worst has been reached, so it behooves us to help in the struggle.

#### EXPERIMENT STILL IN PROGRESS

This study and experiment is being continued, and it is hoped that we may be able to give further facts and details later.

Professor E. R. Lloyd, Dr. Chas. F. Briscoe, and Dr. W. F. Hand have assisted us materially in this experiment, and we are greatly indebted to them. The Parke Davis Company, of Detroit, Michigan, kindly furnished sclerotia of *Claviceps purpurea*.

## SUMMARY

1. *Paspalum dilatatum* is generally considered a valuable forage grass for warm temperate regions.

2. *Paspalum dilatatum* is undesirable as a lawn grass on account of its coarseness and the sticky "Honey-dew" that is formed on diseased heads.

3. *Claviceps Paspali*, *Fusarium heterosporum*, and a species of *Cladosporium* grow on the heads of *Paspalum dilatatum*. The first is a parasite on the grass, while the other two are largely parasites on the first.

4. *Claviceps Paspali* produces numerous sphaelial spores, by means of which the fungus is spread freely to healthy heads by insects that feed on the "Honey-dew" of the fungus.

5. The sclerotia of *Claviceps Paspali* lie on the ground during the winter, and serve to tide the fungus over from one year to the next.

6. Feeding experiments with guinea pigs showed the sclerotia of *Claviceps Paspali* to be poisonous, producing characteristic nervousness and trembling; one gram of the extract produced death in a few hours.

7. The poison in the sclerotia of *Claviceps Paspali* retains its virulence for months after they have been dried; hay containing sclerotia will, apparently, poison animals as readily as the grass.

8. Feeding young or healthy "Paspalum" grass or hay causes no trouble.

9. Feeding "Paspalum" grass or hay infected with *Claviceps Paspali*, and showing sclerotia, or fungus nodules, will result in poisoning in cattle, and, if continued, will cause death.

10. One attack of "Paspalum" poisoning does not produce immunity against a subsequent attack.

11. Cattle seem to acquire a decided appetite for the diseased heads of "Paspalum", and even for the fungus sclerotia.

12. Symptoms seem to indicate the trouble is due to a cumulative poison of some kind.

13. Animals poisoned by "Paspalum" should be taken from the pasture, given a regular dose of epsom or Glauber salts, and a change of feed.

14. "Paspalum" poisoning may be prevented by clipping off the diseased heads of "Paspalum" as often as sclerotia, or fungus nodules, appear; this may be done with mowing machine or hand scythe; one to three mowings during the summer and fall will be necessary.



## REFERENCES

1. Tracy, S. M.;  
Forage Crops for the Cotton Region.  
Farmers' Bulletin 509, U. S. Dept. Agr. (1912).
2. Rolfs, P. H.;  
Grasses, Forage Plants, and Tomato Blight.  
Fla. Expt. Sta. Bul. 18.
3. Wickson, E. J.;  
Distribution of Seeds, Plants, and Scions.  
Cal. Expt. Sta. Bul. 106.
4. Thompson, J. B.;  
Experiments with Field and Forage Crops.  
Guam Sta. Report, 1911.
5. Ritzman, E. G.;  
Forage Crops.  
Porto Rico Sta. Report, 1912.
6. Rhodesia Agricultural Journal 10 (1912) No. 2;  
Dry Land Pastures.
7. Williams, H. M.;  
A Few Observations on *Paspalum Dilatatum*.  
Agr. Gaz. New South Wales 9, pt. 5 (1898).
8. Spillman, W. J.;  
Grasses and Clovers Used in Meadows and Pastures.  
Bailey's Cyc. Amer. Agr., Vol. II., p. 451.
9. Small, J. K.;  
Flora of the Southeastern United States, 1903.
10. Mercier, L.;  
The Role of Insects as Agents in the Propagation of Ergot  
of the Gramineae.  
Comp. Rend. Soc. Biol. 70: (1911), No. 8, pp. 300-302.
11. Stevens, F. L. and Hall, J. G.;  
Three Interesting Species of Claviceps.  
Bot. Gaz. Vol. 50, pp. 460-463.

12. Zimmermann, H.;  
Some Experiments on the Germinative Ability of Old Ergot  
Sclerotia.  
Ztschr. Pflauzenkrank 16: (1906) No. 3, pp. 129-131.
13. Falck, R.;  
On the Air Infection of Ergot and the Dissemination of  
Plant Diseases by Means of Temperature Currents.  
Ztschr. Forst. u. Jagdw, 43: (1911) No. 3, pp. 202-227.
14. Stäger, R.;  
Infection Experiments with Conidia of *Claviceps Purpurea*.  
Mycol. Centbl. 1: (1912) Nos. 7-8.
15. Barnes, B.;  
On the Ergot of Wild and Cultivated Grasses.  
Math. u. Naturw. Ber. Ungarn, 24: (1906) p. 377.
16. Stäger, R.;  
Inoculation Experiments with Grass Infesting Ergots.  
Bot. Centbl. 83: (1900) No. 5.
17. Heald, F. D. and Peters, A. T.;  
Ergot and Ergotism.  
Press Bul. No. 23, Neb. Expt. Sta. (1906).
18. Williams, T. A.;  
Some Plants Injurious to Stock.  
Bul. No. 33, S. D. Expt. Sta. (1893).
19. Pammel, L. H.;  
Manual of Poisonous Plants, pp. 1-977.
20. Salmon, D. E.;  
Enzootics of Ergotism.  
U. S. Dept. Agr. Report, 1884, pp. 212-252.
21. Stalker, M.;  
Ergotism in Cattle.  
Bul. No. 17, Iowa Expt. Sta. (1892).
22. Wilcox, E. V.;  
Ergotism in Horses.  
Bul. 22, Mont. Expt. Sta. (1899).